

Schistosomiasis

Epidemiology:

- Schistosomiasis is known as bilharzia or bilharziasis, after Theodor Bilharz, who first described the cause of urinary schistosomiasis in 1851.
- The disease is found in tropical countries in Africa, the Caribbean, eastern South America, Southeast Asia and in the Middle East

Epidemiology

- Schistosomiasis is endemic in Egypt, exacerbated by the country's dams and irrigation projects along the Nile.
- Infected villagers were treated with repeated shots of tartar emetic. It has been hypothesized that this campaign unintentionally spread the hepatitis C virus via unclean needles.

Classification

Species of Schistosoma that can infect humans:

Schistosoma haematobium causes urinary schistosomiasis

Schistosoma japonicum & Schistosoma mansoni cause intestinal schistosomiasis

Life cycle:

Eggs are eliminated with urine (1). The eggs hatch and release miracidia (2), which swim and penetrate specific snail intermediate hosts (3). The stages in the snail include generations of sporocysts (4) and the production of cercariae (5). The infective cercariae swim, penetrate the skin of the human host (6), and shed their forked tail, becoming schistosomulae (7). The schistosomulae migrate through the veins (8,9). Adult worms in humans reside in the venous plexus of bladder. The females deposit eggs in the small venules of the perivesical systems. The eggs are moved progressively toward the lumen of the bladder and ureters and are eliminated with urine.

Pathogenesis

Bilharziasis is a disease of veins.

Bilharzial ova either cross to the lumina of hollow viscera or entrapped in the submucosa to be: destroyed by the host's granulomatous response or calcified.

B/ granuloma is formed of macrophages, lymphocytes and eosinophils around the eggs.

Clinical stages

3 Stages:

Swimmer's itch (Cercarial penetration)

- **Acute schistosomiasis** (Ova deposition)

Chronic schistosomiasis.

Lesions are controlled by:

The intensity of the infestation, its duration & repetition

- **The host specific response.**

Administration of treatment.

Acute schistosomal lesions

- Perioval **granulomas**: Large bulky, hyperemic masses.
- **Polypoid patches**: mainly in children.
Polyps may obstruct the ureter, or rarely, the bladder outlet.
Terminal hematuria with the passage of ova in urine is due to microscopic abrasions in bladder mucosa.

Chronic schistosomiasis

After some years

Symptoms & Signs are due to the sequels & complications rather than by the infect.

- 30% of light infections resolve spontan.
- Sandy patches: entrapped eggs supplanted by fibrous tissue. (Sand under water)
- Obstructive uropathy: at the sites of intense infestations (Bladder & L. Ureters)
fibrosis leads to BNO &/or Stricture ureter.

Bilharziasis of the urinary bladder

- The Bladder is the commonest organ involved & the heaviest to be affected bec. It is surrounded by pelvic pool of veins.
- The submucosa is the commonest site of ova deposition as it is the most vascular.
- Trapped ova create F.B. reaction which is the cause of pathological lesions in U.B.

Pathological Changes

Hypertrophic or Atrophic

Hypertrophic changes:

Early, common in young children.

F.B. reaction leads to increase vascular supply to mucosa leading to hypertrophy.

Atrophic changes:

Late, Healing of submucosal lesions by fibrosis hinders bl. Supply to mucosa leads to ischemia

Hypertrophic changes

- Nodules, polyps, ulcerating granulomata manifested clinically by repeated attacks of total hematuria. TTT anti B, Laser photocoagulation or TUR.
- Cystitis glandularis: oval gland like structures filled with mucoid secretion. IT is a precancerous lesion.
- Cystitis cystica: Originate from degenerated Von Brunn's nests or cystitis glandularis.
- Leukoplakia: white thick mucosal lesions, progress to squamous metaplasia or S.C.C.

Atrophic changes:

- **Sandy patches**: healed calcified B ova covered by atrophic thin (1-2 epithelial layers) mucosa.
- **Ground glass mucosa**: if atrophy continues, only basement membrane covers increased calcified B ova.

- **Chronic bladder ulcer:** with more atrophy, sloughing of memb. at areas of maximal ischemia occurs. Producing stellate ulcer or fissure ulcer. → periodic postvoiding pain & Bl.

Atrophic changes

- **Calcific plaques:** Localized areas of calcified granulation t. under bladder mucosa.
- **Bladder neck contracture:** becomes rigid and resists funneling during bladder contraction.
- **Contracted bladder:** heavy infestation with severe body reaction → triple pathology (mucosa, submucosal & perivesical tissue)

DIAGNOSIS

I. Clinical manifestations:

Manifestations of acute schistosomiasis:

Terminal hematuria & increased frequency

Painful micturition.

B-Manifestations of ch. B & its complications:

- Terminal & sometimes total hematuria.
- B.M., Postvoiding pain or suprapubic dull aching pain.
- Severe urethral pain during and after voiding.
- Increased frequency and urgency.
- Obstruction due to polyps near B.N. or ureteric orifice.
- Stone formation, hydronephrosis, reflux.

II. Laboratory investigations

- **Urine analysis:** Microscopic hematuria + terminally spined eggs means active disease.
- **Circulating antigen in urine and serum:** In chronic inactive cases (egg excretion is uncommon), serologic, radiologic & cystoscopic diagnosis are superior. Also in monitoring therapy.

III. Radiological investigations

1. Abdominal ultrasonography:

- Focal thickening of bladder wall.
- Large polypoid masses of U.T.
- Hydronephrosis and hydronephrosis
- Associated abdominal pathology e.g. liver cirrhosis, splenomegaly.

2. KUB film:

- Bilharzial calcifications: linear or patchy. Or lower end ureteric calcification.
- 2ry radio-opaque stones.

3. Intravenous urography

- Hydronephrosis and hydronephrosis due to vesico-ureteral reflux or ureteral strictures.
- Filling defects in cystogram.
- Contracted bladder.
- B.N.O. with bladder diverticula and high postvoiding urine volume.

Treatment

I-Medical management:

- **Praziquantel Tab.:** The drug of choice based on its spectrum of activity, its safety and cost. Given as 2 oral doses of 40 mg/kg in 1 day.
- **Mirazid capsules:** 3 oral doses

II- Endoscopic management

- Transurethral resection (TUR) of bilharzial bladder lesions as granuloma, polyp, ulcer or leukoplakia.
- Follow up of leukoplakia as it is precancerous
- Any suspicious lesion or neoplasm should be biopsied.

III- Surgical management

For lesions not responding to med. TTT

- Partial cystectomy: for ch. B. ulcer, polyp, or large granulomata.
- Augmentation ileal cystoplasty: for contracted bladder.
- Radical cystectomy with suitable diversion for bladder cancer.

Bilharziasis of the ureter

- Commonly affects the lower thirds.
- Less commonly; affects the Lt. ureter at the level of L3 T₁₂ process (Site of porto-systemic anastomosis).
- Healing leads to stricture due to fibrosis with subsequent hydrouretero-nephrosis.
- KUB → linear calcification or 2 stones.
- IVU or MRU → degree of hydroureter.
- VCUG → detect vesicoureteric reflux.
- Ureterscopy to dilate and stent ureteral stricture.
- Surgery: ureteroneocystostomy or Boari flap.

Other sites for bilharzial affection

- The prostate, seminal vesicles & bulbar urethra rarely affected.
- Prostate: nodular or DRE.
- Seminal vesicles: Calcified S.V → honey-comb appearance on KUB film.
- Urethral mass may fistulate into the perineum (Water cane fistula) without urethral narrowing.
- Spermatic cords: show military, nodular or massive affection.

GOOD LUCK

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